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Niwarinda, 2025
ISSN: 2579-0781

International Digital Organization for Scientific Research
IDOSR JOURNAL OF EXPERIMENTAL SCIENCES 11(2): 78-88, 2025.
https://doi.org/10.59298/IDOSR/JES/112.7888
IDOSRJES11200000

Natural Product-Derived Modulators of the Gut Microbiota in the Prevention of Obesity-Induced Diabetes

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ABSTRACT

The global prevalence of obesity and type 2 diabetes mellitus (T2DM) continues to rise at an alarming rate, posing a major public health and economic challenge worldwide. Growing evidence indicates that the gut microbiota plays a crucial role in the development of obesity-induced diabetes through mechanisms involving energy homeostasis, lipid metabolism, glucose regulation, and systemic inflammation. Dysbiosis, characterized by an imbalance in the gut microbial community, contributes significantly to metabolic dysfunction and insulin resistance. Natural products, particularly those derived from plants, herbs, and dietary sources, have emerged as promising modulators of the gut microbiota with therapeutic potential in preventing and managing obesity-induced diabetes. These bioactive compounds, including polyphenols, dietary fibers, alkaloids, saponins, and prebiotics, can restore microbial balance, enhance short-chain fatty acid production, reduce endotoxemia, and modulate host metabolism. This review provides a comprehensive overview of the interplay between gut microbiota and metabolic diseases, highlights natural product-derived interventions for gut microbiota modulation, and discusses the mechanistic insights and therapeutic prospects of targeting the gut microbiota in the prevention of obesity-induced diabetes.

Keywords: Gut microbiota, Natural products, Obesity, Type 2 diabetes mellitus, Dysbiosis, Metabolic disorders

INTRODUCTION

Obesity and type 2 diabetes mellitus (T2DM) are interlinked metabolic disorders that have reached epidemic proportions globally, creating an urgent need for effective prevention and management strategies[1–3]. According to the World Health Organization, obesity has nearly tripled since 1975, with over 650 million adults classified as obese, while an estimated 537 million people worldwide are currently living with diabetes, most of whom have T2DM[4–6]. The coexistence of these conditions, commonly referred to as "diabesity," not only elevates the risk of cardiovascular diseases and certain cancers but also severely impacts life expectancy and quality of life[7, 8]. Despite advances in pharmacotherapy, including insulin analogs, GLP-1 receptor agonists, and SGLT2 inhibitors, long-term management remains challenging due to limited accessibility, cost, and adverse side effects[9, 10]. Consequently, attention has shifted toward alternative and complementary strategies rooted in nutrition, lifestyle, and natural product interventions.

Over the past two decades, the gut microbiota has emerged as a central player in the pathogenesis of obesity and diabetes. The human gastrointestinal tract harbors trillions of microbes, including bacteria, viruses, fungi, and archaea, whose collective genome, known as the microbiome, exerts profound influence on host physiology[11–13]. A healthy gut microbiota contributes to nutrient metabolism, vitamin synthesis, maintenance of gut barrier integrity, and modulation of the immune system. However, disruptions in the microbial community, referred to as dysbiosis, have been strongly associated with obesity, insulin resistance, and T2DM[14, 15]. Dysbiosis often manifests as a reduction in microbial diversity, an altered Firmicutes-to-Bacteroidetes ratio, and increased abundance of pathogenic species that produce endotoxins such as lipopolysaccharides (LPS). These microbial shifts can promote systemic low-grade inflammation, impair insulin signaling, and disrupt metabolic homeostasis[14, 16, 17].

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Several mechanistic pathways link gut microbiota dysbiosis with obesity-induced diabetes. One key mechanism involves energy harvest and storage[18, 19]. Certain gut microbes possess specialized enzymes that allow efficient fermentation of otherwise indigestible dietary polysaccharides, producing short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate. While SCFAs are generally beneficial in maintaining colonic health and regulating glucose metabolism, excessive SCFA production has been implicated in increased adiposity due to enhanced energy extraction[20, 21]. Moreover, dysbiosis-induced changes in SCFA composition may disrupt the balance between lipogenesis and fatty acid oxidation. Another critical pathway involves metabolic endotoxemia[22, 23]. Elevated levels of circulating LPS derived from Gram-negative bacteria can trigger Toll-like receptor 4 (TLR4)-mediated inflammatory responses, leading to chronic low-grade inflammation, a hallmark of insulin resistance and T2DM. Additionally, gut microbiota influence bile acid metabolism, incretin secretion, and the gut–brain axis, further impacting energy balance and glucose regulation[24, 25].

Given this intricate relationship between gut microbiota and metabolic diseases, restoring microbial balance has become an attractive therapeutic strategy. Traditional pharmacological agents, while effective in controlling glycemia, do not directly address the underlying microbial dysfunction. This gap has spurred research into dietary and natural product-derived interventions as safe, cost-effective, and sustainable alternatives. Natural products, encompassing plant-derived compounds, functional foods, and bioactive metabolites, have shown remarkable potential in modulating the gut microbiota. Polyphenols, found abundantly in fruits, vegetables, tea, coffee, and wine, are poorly absorbed in the small intestine, allowing significant quantities to reach the colon where they interact with gut microbes[26, 27]. These compounds exert prebiotic-like effects by promoting the growth of beneficial bacteria such as Bifidobacteria and Lactobacilli, while suppressing pathogenic species. Similarly, dietary fibers serve as substrates for microbial fermentation, enhancing SCFA production and improving insulin sensitivity. Other natural products such as alkaloids, terpenoids, saponins, and polysaccharides have demonstrated microbiota-modulating properties, often accompanied by anti-inflammatory and antioxidant effects that protect against metabolic dysfunction[28, 29].

Clinical and preclinical studies provide accumulating evidence supporting the efficacy of natural product-derived microbiota modulators in preventing obesity-induced diabetes. For instance, resveratrol, a polyphenolic compound from grapes, has been shown to reverse high-fat diet-induced dysbiosis and improve glucose tolerance in animal models[30]. Curcumin, derived from turmeric, not only enhances microbial diversity but also reduces systemic inflammation and oxidative stress associated with T2DM [31]. Probiotics and prebiotics derived from natural sources further contribute by replenishing beneficial microbial populations and restoring intestinal barrier integrity[32–35]. The therapeutic potential of these natural products lies not only in their direct metabolic effects but also in their capacity to synergize with the host microbiota to restore homeostasis. In this review, we critically examine the current understanding of gut microbiota modulation by natural products and its implications for the prevention of obesity-induced diabetes. We begin by exploring the pathophysiological links between gut microbiota dysbiosis and metabolic disorders. We then highlight various classes of natural products with demonstrated microbiota-modulating potential, emphasizing their mechanisms of action, preclinical and clinical evidence, and translational prospects. Finally, we discuss emerging challenges and future directions in harnessing natural products as microbiota-targeted interventions for combating diabesity.

2. Gut Microbiota and the Pathophysiology of Obesity-Induced Diabetes

The gut microbiota plays a pivotal role in shaping host metabolic processes, and disturbances in its composition and function have been strongly implicated in obesity-induced diabetes[18, 22, 30]. The microbial ecosystem of the gut consists predominantly of Firmicutes and Bacteroidetes, with smaller proportions of Actinobacteria, Proteobacteria, and Verrucomicrobia. An imbalance in these populations is frequently observed in obese and diabetic individuals, with an increase in Firmicutes relative to Bacteroidetes often linked to enhanced energy harvest and adiposity[36–38]. This altered ratio enhances the capacity of the gut microbiome to extract calories from indigestible polysaccharides, resulting in excess storage of fat and disruption of energy balance.

Another key pathogenic mechanism involves metabolic endotoxemia. Lipopolysaccharides (LPS), components of the outer membrane of Gram-negative bacteria, can translocate across the intestinal barrier when dysbiosis weakens gut integrity[39, 40]. Elevated LPS in the systemic circulation activates Toll-like receptor 4 (TLR4) on immune cells, initiating pro-inflammatory cascades and the release of cytokines such as TNF-α and IL-6. This chronic low-grade inflammation directly contributes to insulin resistance by impairing insulin receptor signaling pathways [41]. Moreover, gut permeability is further compromised by decreased expression of tight junction proteins, which facilitates bacterial translocation and exacerbates systemic inflammation[42, 43].

Bile acid metabolism is another crucial link between gut microbiota and host metabolic health. Gut microbes convert primary bile acids into secondary bile acids, which serve as signaling molecules for receptors such as farnesoid X receptor (FXR) and G-protein coupled bile acid receptor (TGR5). These receptors regulate lipid metabolism, glucose homeostasis, and energy expenditure[44]. Dysbiosis-induced alterations in bile acid profiles can impair these regulatory mechanisms, contributing to glucose intolerance and weight gain.

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Short-chain fatty acids (SCFAs), primarily acetate, propionate, and butyrate, are beneficial microbial metabolites produced from the fermentation of dietary fibers. SCFAs influence host metabolism by enhancing gut hormone secretion, improving insulin sensitivity, and maintaining colonic epithelial integrity[21, 23, 45]. However, dysbiosis can lead to an imbalance in SCFA production, disrupting glucose and lipid metabolism. Additionally, the gut microbiota communicates with the central nervous system via the gut-brain axis, influencing appetite regulation and energy intake. Disruptions in this signaling pathway have been associated with overeating, obesity, and impaired glucose regulation[46, 47].

Collectively, these mechanisms highlight the central role of the gut microbiota in obesity-induced diabetes. Understanding these pathways provides a strong rationale for microbiota-targeted interventions, particularly those derived from natural products, as a preventive and therapeutic strategy against diabesity.

3. Natural Products as Modulators of Gut Microbiota

Natural products represent a diverse reservoir of bioactive compounds that exert profound effects on gut microbial composition and activity, offering promising therapeutic potential in the prevention of obesity-induced diabetes[48, 49]. Unlike synthetic drugs, many natural products act gently yet effectively, producing multifaceted benefits that include restoring microbial diversity, promoting the growth of beneficial species, and suppressing opportunistic pathogens. Among these, dietary polyphenols, dietary fibers, alkaloids, saponins, polysaccharides, and terpenoids have received significant attention for their microbiota-modulating properties[50–52].

Polyphenols, abundant in fruits, vegetables, tea, coffee, cocoa, and wine, are poorly absorbed in the small intestine, allowing substantial quantities to reach the colon, where they undergo microbial biotransformation[36, 53, 54]. These interactions not only alter microbial composition but also produce bioactive metabolites with enhanced biological activities. For instance, polyphenols promote the growth of Bifidobacteria and Lactobacilli while inhibiting pathogenic bacteria such as Clostridium perfringens. This modulation enhances intestinal barrier integrity, reduces endotoxemia, and improves glucose tolerance. Similarly, dietary fibers act as prebiotics by serving as substrates for microbial fermentation, leading to increased production of short-chain fatty acids (SCFAs)[21]. SCFAs improve host metabolism by enhancing satiety hormones such as GLP-1 and PYY, improving insulin sensitivity, and regulating lipid metabolism.

Other natural compounds, such as alkaloids and saponins, have also demonstrated microbiota-modulating effects[55]. Berberine, an isoquinoline alkaloid, has been shown to restructure microbial communities, increasing Akkermansia muciniphila, a bacterium associated with improved insulin sensitivity and reduced adiposity[56–58]. Saponins from plants like ginseng not only exert anti-inflammatory effects but also reshape gut microbiota to favor beneficial species[59, 60]. Additionally, polysaccharides from mushrooms, seaweeds, and legumes act as potent prebiotics, stimulating SCFA production and modulating immune responses. Terpenoids, including limonene and curcuminoids, contribute by reducing systemic inflammation and fostering microbial diversity[61].

Importantly, the interaction between natural products and gut microbiota is bidirectional. While natural compounds influence microbial composition, microbes metabolize these compounds into bioactive derivatives that exert systemic effects on host metabolism. This synergistic relationship underpins the therapeutic potential of natural product interventions. Preclinical and clinical evidence consistently supports that natural product-derived modulation of the gut microbiota improves glucose metabolism, reduces weight gain, and protects against insulin resistance[62, 63]. These findings collectively underscore the promise of natural products as safe, sustainable, and effective tools to prevent and manage obesity-induced diabetes through targeted modulation of the gut microbiota.

4. Mechanistic Insights into Gut Microbiota Modulation by Natural Products

The mechanistic basis for how natural products modulate gut microbiota and confer protection against obesity-induced diabetes lies in the interplay between microbial metabolism, host signaling pathways, and systemic metabolic regulation. One central mechanism involves the enhancement of short-chain fatty acid (SCFA) production[64, 65]. When dietary fibers and polysaccharides from natural sources are fermented by gut microbes, SCFAs such as acetate, propionate, and butyrate are produced. These metabolites act as signaling molecules, binding to G-protein coupled receptors such as GPR41 and GPR43, thereby promoting the secretion of satiety hormones like peptide YY (PYY) and glucagon-like peptide-1 (GLP-1). This leads to reduced appetite, improved insulin sensitivity, and enhanced glucose homeostasis[66]. Butyrate, in particular, is critical for maintaining gut barrier integrity, reducing endotoxemia, and mitigating chronic inflammation, all of which are implicated in obesity-induced diabetes[67].

Another key mechanism involves modulation of bile acid metabolism. Natural products influence gut microbial enzymes such as bile salt hydrolases, which convert primary bile acids into secondary bile acids. These metabolites interact with nuclear receptors such as the farnesoid X receptor (FXR) and G-protein coupled bile acid receptor (TGR5). Activation of these receptors regulates lipid metabolism, insulin sensitivity, and energy expenditure, highlighting a direct link between microbial alterations and systemic metabolic health[68, 69].

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Polyphenols, for example, have been shown to modulate bile acid pools and receptor signaling, contributing to improved glucose regulation[70-76].
Natural products also play a role in attenuating metabolic endotoxemia. Dysbiosis often increases Gram-negative bacterial populations that release lipopolysaccharides (LPS). Compounds such as berberine, curcumin, and resveratrol enhance the abundance of beneficial bacteria like Akkermansia muciniphila and Bifidobacteria, which strengthen the intestinal epithelial barrier and reduce LPS translocation[71]. This mitigates chronic low-grade inflammation mediated through Toll-like receptor 4 (TLR4) and downstream NF-κB pathways, ultimately improving insulin sensitivity[77-83].
Furthermore, natural products influence host redox balance and immune modulation through microbiota interactions. For instance, flavonoids and phenolic acids not only undergo microbial transformation into bioactive metabolites but also exert antioxidant and anti-inflammatory effects that protect pancreatic β-cells and improve glucose tolerance[84-89]. Probiotic strains stimulated by prebiotic fibers or plant polysaccharides contribute additional immune-regulatory effects by modulating T-regulatory cells and cytokine production.
Another emerging mechanism is the modulation of the gut–brain axis. SCFAs and microbial metabolites derived from natural products influence vagal nerve signaling and neurotransmitter production, which affect appetite regulation and energy intake. This neural integration underscores how gut microbiota modulation extends beyond local intestinal effects to systemic metabolic and neuroendocrine regulation.
Together, these mechanistic insights illustrate that natural product-derived interventions act not only through direct bioactivity but also via their capacity to reshape the gut microbiota, leading to systemic metabolic benefits crucial for the prevention of obesity-induced diabetes.

5. Clinical Evidence and Translational Potential

While much of the evidence regarding natural product-derived modulation of the gut microbiota in obesity-induced diabetes stems from animal models, a growing body of clinical research is validating these findings in human populations. Several randomized controlled trials (RCTs) have demonstrated that polyphenol-rich foods, dietary fibers, and prebiotics can beneficially modulate the gut microbiota, leading to measurable improvements in metabolic health outcomes[90-97].
For example, clinical studies on polyphenol-rich green tea and grape products have reported enhanced microbial diversity, increases in Bifidobacterium and Lactobacillus populations, and improvements in insulin sensitivity among overweight and obese individuals. Resveratrol supplementation in humans has been associated with reduced markers of systemic inflammation, improved lipid profiles, and favorable changes in microbial composition[98-103]. Similarly, curcumin has shown promise in clinical settings, where it improves glycemic control and reduces inflammatory biomarkers, effects that appear partially mediated by shifts in gut microbial communities.
Dietary fiber interventions have been particularly well-documented. Supplementation with inulin, oligofructose, or resistant starch has consistently increased SCFA production, reduced body weight, and improved glycemic control in both diabetic and prediabetic subjects. These outcomes are often accompanied by increased abundances of SCFA-producing bacteria such as Faecalibacterium prausnitzii and Roseburia spp[104-110]. Clinical studies with polysaccharides from mushrooms, seaweeds, and legumes also highlight their prebiotic properties and ability to improve glucose metabolism.
Probiotics and synbiotics (combinations of probiotics and prebiotics) have also been investigated in clinical trials targeting obesity and diabetes. Specific strains such as Lactobacillus rhamnosus and Bifidobacterium animalis have demonstrated improvements in insulin sensitivity and reductions in fasting glucose[111-119]. When combined with prebiotic fibers, synbiotics enhance microbial stability and lead to more pronounced metabolic benefits.
Despite these encouraging findings, challenges remain in translating microbiota-targeted interventions into widespread clinical practice. Inter-individual variability in microbiota composition means that responses to natural product interventions can differ significantly between individuals. Moreover, the bioavailability and pharmacokinetics of many natural products are limited, raising questions about optimal dosing strategies[120-124]. Long-term safety and efficacy also need to be established through large-scale, multicenter clinical trials.
Nevertheless, the translational potential is substantial. Natural products are generally safe, affordable, and culturally acceptable, making them attractive for population-level interventions. Personalized nutrition strategies that combine microbiome profiling with targeted natural product interventions may represent the next frontier in the prevention of obesity-induced diabetes [125-133].

6. Future Perspectives

The intersection of natural products, gut microbiota, and metabolic health represents a rapidly expanding frontier in biomedical research. While considerable progress has been made in elucidating the role of natural product-derived microbiota modulators in preventing obesity-induced diabetes, future research must address key gaps to fully harness their therapeutic potential.
One major area for advancement is the personalization of microbiota-targeted therapies. Since gut microbial composition varies widely between individuals due to genetics, diet, environment, and lifestyle, a "one-size-fits-

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all" approach may not yield consistent results. Integration of microbiome sequencing, metabolomics, and computational modeling could facilitate precision nutrition strategies that tailor natural product interventions to individual microbial profiles. This personalized approach may maximize efficacy while minimizing variability in clinical outcomes.

Another pressing need is the standardization of natural product formulations and dosing regimens. The bioavailability of polyphenols, alkaloids, and other bioactives is often limited, and their metabolic effects depend on interactions with gut microbes. Novel delivery systems such as encapsulation, nanoformulations, and fermentation-based processing may enhance bioavailability and ensure consistent therapeutic outcomes. In addition, future clinical trials must adopt rigorous designs with adequate sample sizes, longer intervention periods, and standardized endpoints to validate the efficacy and safety of these interventions.

The integration of natural product-derived microbiota modulators with existing therapeutic strategies also holds promise. Combining dietary interventions with pharmacological agents or lifestyle modifications such as exercise may produce synergistic effects in preventing or managing obesity-induced diabetes. Importantly, regulatory frameworks and public health policies must evolve to recognize the therapeutic value of natural products, ensuring quality control and promoting accessibility in both developed and developing nations.

CONCLUSION

In conclusion, natural product-derived modulators of the gut microbiota represent a compelling and sustainable strategy for preventing obesity-induced diabetes. Through mechanisms involving SCFA production, bile acid metabolism, attenuation of endotoxemia, and modulation of the gut–brain axis, these bioactives restore microbial homeostasis and confer systemic metabolic benefits. Clinical evidence, though still emerging, supports their translational potential, particularly when integrated into personalized and holistic healthcare strategies. As research advances, the synergistic relationship between natural products and the gut microbiota may provide the foundation for innovative interventions aimed at curbing the global diabesity epidemic.

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CITE AS: Niwarinda Arnold (2025). Natural Product-Derived Modulators of the Gut Microbiota in the Prevention of Obesity-Induced Diabetes. IDOSR JOURNAL OF EXPERIMENTAL SCIENCES 11(2): 78-88. https://doi.org/10.59298/IDOSR/JES/112.7888

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